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A STUDY OF OCULAR COLOBOMA.

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WHILE instances of coloboma of one or more coats of the eyeball are not among the extreme scarceties in ocular anomalies, the unsettled state of opinion as to their nature and causation warrants the record of any example that promises to throw some additional light on the question of their origin. This is my justification for a report and study of the following cases:

T. W., aged 40 years, came to my clinic at the Emergency Hospital on account of an injury to the left eye, caused by the explosion of a fire-cracker on the 4th of July, 1897. A grain of powder had passed through the cornea and lodged in the iris, and there were several grains in the conjunctiva. A glance showed the existence of a downward coloboma in both irides and led to a detailed examination of both eyes as to the conditions which are here reported. Of the injury, it will suffice to say, that under treatment it went on to recovery leaving a posterior synechia of the upper edge of the pupil to the lens capsule, and a slight corneal opacity.

RIGHT EYE.—The *coloboma of the iris* is represented in

Fig. 1, the pupil being under the influence of atropia. There is a thin streak of opacity in the lens just at the seat of the coloboma and near the margin. Whether or not this opacity is congenital it is impossible, now, to tell, but as there is no history of trauma, and there are no spiculæ of opacity elsewhere in this lens or in the other, the inference is that it is. The vision is $^{5}/_{xxxv}$, but with — 1 at 75° it is increased to $^{5}/_{xxx}$. This has always been his worse eye. The cornea, examined by the ophthalmometer, was found to be within the normal curvature having 41.5 D. at 75° and 42 5° at 135°, at the point of fixation, showing, however, a departure as to the relative direction of the axis of the meridians, due no doubt to a large angle alpha.

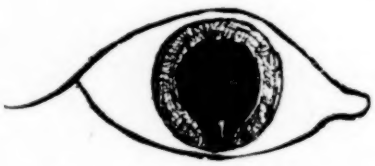


FIG. 1.– Coloboma of iris, with slight lenticular opacity. Right eye of T. W.

The ophthalmoscope revealed the appearances shown in Fig. 2. This drawing represents with very considerable accuracy the topography of the back ground, being the outcome of numerous sittings and many careful sketches. A first inspection shows a very bewildering collection of vessels running in all directions, seemingly on no definite plan and greatly at variance with the usual system of distribution. A careful study, however, will soon extract some order out of the chaos, and a modification—though a very extensive one—of the regular vascular distribution can be made out.

A coloboma of the choroid stretches from the margin of the optic nerve entrance as far forward as can be seen, and undoubtedly extends through the ciliary body to meet the coloboma of the iris which is in direct line with it. It also, apparently, passes up beyond the nerve head, since the exit of the retinal vessels seems to be far below the upper border of the defect. What must be considered as the lower edge of the disc lies as much as three ordinary disc-diameters below the upper edge of the defect. (The drawings are made from the

inverted image, but will be referred to in their normal relations as seen in the direct image). The lower edge of the disc is quite clearly defined.

FIG. 2.—Fundus of right eye. T. W. A, the arteries; B, large tortuous vein possibly choroidal; C, choroidal veins; D, vessel at edge of disc; E, Varicose retinal vein. — 3 and — 10 number of concave glass required to see the fundus at that portion of the eye-ground.

The retinal vessels coming up to its edge and dropping down with a short curve disappear under it. In most cases it is possible to distinguish the arteries (A) from the veins (V) by their straight course and smaller caliber. It will be noticed (and this is the case with the other eye, Fig. 5), that the arteries are much thinner than usual, whereas the veins are of about normal size. A careful analysis will reveal the presence of what, in the normal distribution, would be a superior and inferior, one nasal and two temporal arteries, while one vessel running down and out and which is very tortuous at the beginning is doubtful as to classification. The same is true of some fine vessels springing from the lower edge of the disc and running outwards for a short distance. One vessel, marked (B), is also difficult to classify. It starts from among the fibers of the

disc not far from the upper edge, is very tortuous in its course and ends abruptly at the choroidal edge in a dense mass of pigment. Another peculiarity of the vessel is that it lies deeper than any of the other vessels, requiring a — 17 to be seen distinctly at its point of origin. The one marked D also starts deeply and curves round what seems to be the edge of the disc, losing itself in a fine point, but evidently giving off some of the vessels which run laterally. The vessels marked C have very indistinct outlines and must be regarded as choroidal, since they have no connection with the retinal system. At E we have a very large, almost varicose, branch of a retinal vein which disappears at the edge of the choroid. In no other portion of the area of the coloboma are there any apparent remnants of choroidal structure. The edge however is strongly pigmented in places. Starting from the lower edge of the disc there are dark band-like areas with edges sharply defined at the beginning but gradually losing this definition to become diffused, going upward and outward toward the upper border of the disc which, however, can not be clearly outlined. This area, it seems to me, is the optic disc very much stretched and elongated backward. That the main portion of the disc is displaced below its normal position, is evidenced by the fact that the macula lutea, as indicated by the point of fixation, is about on a level with the exit of the upper choroidal vein, while the exit of the lower veins at the sharp edge of the disc is at least one disc diameter and a half below the macula. The extension of the coloboma above the optic nerve entrance is therefore more apparent than real, the upper edge of the choroidal defect no doubt representing approximately the upper edge of the disc. The whole picture suggests the appearance as if the optic nerve had been seized by its lower edge and pulled strongly downward, the upper edge remaining stationary, as shown in Fig. 3, where A represents the lower edge of the optic nerve in its normal position pulled backward and downward to B. This represents quite well the appearances which are usually designated "coloboma of the optic nerve and optic nerve sheath." We shall see, however, that it does not at all necessarily signify a defect in the continuity of either of those structures. The surface of the defect is not all of the same radius of curvature, some portions lying much behind the others. At the upper edge of the coloboma of the choroid the refraction

as determined by the ophthalmoscope is emmetropic; some distance below it is — 3, and below the lower edge of the stretched disc it is — 10. The shadow test applied to different parts of the coloboma shows emmetropia at or near the upper part of the defect passing over to — 12 for the anterior part as far forward as it could be applied. All this shows conclusively that the optic nerve and the tissues of the fundus at this part had been pushed back as indicated in the Fig. 3.

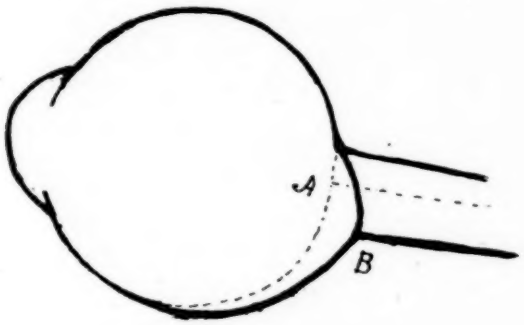


FIG. 3.—Stretching of the back of the eyeground, and especially the the nerve head. from pressure. The nerve head, A, is dragged back and stretched until it assumes the position and form shown at B.

The *visual field* is given in Fig. 4 and shows limitations corresponding pretty accurately with the defect in the back-

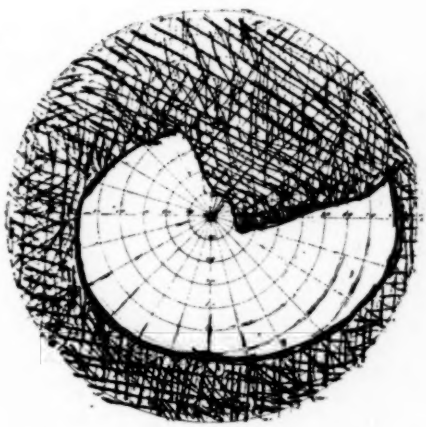


FIG. 4.— Visual field of right eye.

ground. It will be noticed that the defect to the temporal side passes below the point of fixation at a place corresponding to the locality of the optic nerve entrance, caused, no doubt, by an extension of the normal blind spot and due to the anatomical conditions revealed in Fig. 2.

FIG. 5.—Fundus of left eye. V, choroidal vein; A, remnants of choroidal stroma; B, atrophic spot in the choroid.

LEFT EYE.—The *iris coloboma* is the counterpart of that of the right. The *choroidal coloboma* is shown in Fig. 5. As will be seen this differs in several particulars from that of the right and this is especially noticeable as regards the optic nerve. The *nerve head* here assumes the form of a horizontal oval, which it is commonly represented as having in the typical pictures of coloboma of the optic nerve and optic nerve sheath seen in the atlases. It is of a dull color without any tinge of red and its scheme of vascularization differs widely from the normal. From near the center of the coloboma there springs a large vessel (V) which runs some distance, then bifurcates and one branch disappears at the choroidal edge apparently entering the choroid. It evidently belongs to the choroidal and not to the retinal system of vessels. In its immediate vicinity

there are two spots (A, A), which are evidently the remains of the choroidal stroma. In this eye, as in the other, the arteries are of abnormally small calibre. To the upper and inner quadrant (lower and outer in the figure) of the disc there is a spot of choroidal atrophy about half the size of the disk, but separated from it by apparently normal tissue. On the surface of this spot there are remains of vessels and choroidal stroma. The rest of the fundus does not depart from the normal. The *visual field* is given in Fig. 6. and shows the same dip down

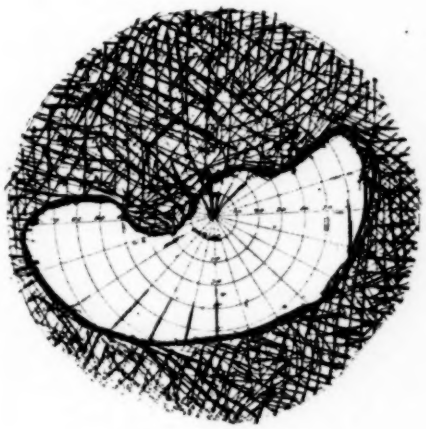


FIG. 6. - Visual field of left eye.

below the point of fixation we find in the other eye. There is, however, a greater restriction down and out than in the right eye. On account of the iritic adhesions and the corneal opacity,—results of the injury,—it was not so easy to make exact measurements of the depth of the coloboma, but it was determined that the upper edge of the disc was emmetropic, while it required a — 3 to see the lower edge. In the area of the coloboma itself the vessels were not sufficiently well outlined to enable even an approximate estimate of their position to be made. The shadow-test, however, revealed a high degree of myopia in that region of the background. V. = $^{5}/_{XXX}$, notwithstanding a considerable opacity of the cornea which gave rise to quite an amount of irregular astigmatism as revealed by the ophthalmometer. Before the accident this was his better eye and with it he could read without difficulty.

As regards *family history*, he states that his father had a slit like his own in both eyes, and that his vision was always bad; that a brother had a slit in the iris of the left eye, and that the iris in this eye was black, while that of the other was blue. His own irides are blue.

It is difficult to reconcile fully the appearances found in most cases of coloboma oculi with the manner of development of the eye at present recognized by embryologists, and the cause that has by almost common consent been assigned for them—namely, a failure of the fœtal cleft to close.

As regards the *iris* it is to be remembered that the whole anterior part of the uveal tract—iris, ciliary body and processes, are developed only in part from the choroidal portion of the enveloping mesoderm. The inner and pigment layers of the iris are developed from the retinal layer and not from the choroidal layer, the latter furnishing the stroma and blood vessels. The development of the iris does not commence before the beginning of the third month, and some time after the closure of the fœtal cleft, and the fœtal cleft while it reaches, does not involve the region from which the iris is developed. Moreover, there is at no time during the development of the iris any thing resembling a cleft. It is developed uniformly in all directions from the periphery towards the center. As, however, the interior layers of the iris are developed from the retinal layer there would seem to be some justification for a coloboma iridis being connected in some way with a genuine non-closure of the cleft, when one existed. As, however, there is lacking in such cases any convincing evidence of a failure of the cleft to unite we must seek other causes. It seems most probable, in the light of the facts now in our possession, that the origin of coloboma of the iris is connected in some way with an interference with the blood supply as first pointed out by Arnold in 1838. It seems almost certain, too, as suggested by Posey, in a recent paper on the subject, that the iris is developed in segments, rather than in a continuous circle, and an interference with the development of one of those segments,—it may be through a faulty vascular supply,—would be likely to lead to some one of the iritic anomalies such as coloboma, partial or complete correctopia, or some of the lesser anomalies, such as decoloration. What lends plausibility to this idea is the fact that these anomalies are found at any segmental part of the iris.

We have coloboma of the iris downward, upward, outward, inward or obliquely in any of these directions, and the pupil may be shifted in almost any direction from the center. Some instances of early arrest of development on the part of the iris substantiate this view. In 1875 I reported in *Knapp's Archives* (Vol. IV) a case of rudimentary iris in which this segmental character of development is clearly shown (Fig. 7). Here it would appear all the segments were arrested very nearly equally and at the same time. If, however, the arrest had been limited to any one segment we would have the necessary conditions for a coloboma iridis and it need not necessarily be downward in the direction of the fœtal cleft. It will be noted that there is also in the case (which was bilateral) a central opacity of the lens capsule. In respect to the *choroid*, however, it is easy to see how this theory has some apparent foun-

FIG. 7.—Rudimentary iris, showing the segmental development of the iris — ; opacity at center of the lens.

dation in the facts. Colobomata of the choroid occupy, with very rare exceptions, the position of the fœtal cleft, and the defect is of such a character as to indicate its congenital origin and point to some defect in development. The non-closure of the fœtal cleft, therefore, at first blush, offers itself as the readiest solution of the anomalous condition.

In order to a clear understanding of how far this may be true let us briefly review the most recent and accepted accounts of the method in which the eye is developed. The optic stalk springs from the cerebral vesicle as a hollow tube and on its end is formed the primary optic vesicle. This vesicle is invaginated by the mesodermal layer from below (after the lens has made its way in from above) pushing the wall of the vesicle before it until the cavity is obliterated by contact of its own opposing walls. The cavity is now filled with the mesoderm, which also extends itself around the exterior of the primary vesicle, holding the walls of the primary vesicle, now one mem-

brane, between its interior and exterior portions and forming the secondary optic vesicle. The interior and exterior portions of the mesodermal structure are connected through the space left in the primary optic vesicle when the mesoderm invaginated it. This space is called the *fœtal* or *choroidal cleft.* In time this space is covered over by the development and extension of the walls of the primary optic vesicle and becomes a part of the *retina.* The mesoderm which remains in the interior of the vesicle becomes the *vitreous,* and the outer mesodermal structures, which surround the primary vesicle, form the *choroid* and *sclera.* The cleft in the primary vesicle is usually closed by the end of the second month.

It necessarily follows, if this be true, that a failure of the fœtal cleft to close must affect, primarily, the retina, since this is the only tissue in which there is a discontinuity of tissue. There is not, nor can there be, a cleft in the mesoderm, for it is continuous through the cleft interiorly and exteriorly, and the exterior portion, forming the sclera and choroid is continuous without a break. Whatever defects of structure may follow from abnormal or arrested development at this place, an absence or serious defect in the retina must be the first and a necessary one. It is possible that the mesoderm at the place of intrusion may from some cause fail in proper development and of course then some abnormal condition must result; but if the processes are so far normal as to bring about a perfect formation of the sclera there seems no just reason why the choroid, developing from the same tissue, should be entirely lacking. In fact, a failure of the fœtal cleft to close should carry with it an absence, at that locality, of both sclera and choroid. Instances of this, no doubt, are those cases of so-called anophthalmos or microphthalmos in which a large cyst occupies the place of the eye or springs from its undeveloped structures. This is most likely due to a failure of the cleft to close or to a failure of the mesoderm to invaginate the primary optic vesicle, with a misdirection of the developmental processes. But the normal development having gone on to the formation of the sclera, which virtually completes a closure of the fœtal cleft, it is hard to see how any conditions found in the choroid, which is developed from the same material as the sclera, can be referred to a non-closure of the cleft. It is admitted, however, that this region, which is the meeting place of

several developmental forces, is more likely than elsewhere to be the seat of abnormalities of which a failure of the cleft to close is only one and probably a rare one, and would render it more liable to arrests or deviations in development or pathologic processes of various kinds.

That these anomalies are due for the most part to arrest or a defect in development is evident from their appearance. This is shown in the character of the vessels—the smallness of the arteries, and tortuosity of the veins and the peculiarity in their distribution; also in the appearance of the disc in some cases where the abnormality has extended to that tissue. Of course these might be, but it is hardly possible that they are, the results of an inflammation *in utero*. They have all the features of congenital anomalies, the result of defective development. There are other appearances, however, which are very like the changes we should expect from inflammatory processes and their results. The edges of the coloboma are, as a rule, heavily pigmented, at least at places, and there are remnants of choroidal stroma and pigment within the area of the coloboma, and it is very common to find choroidal vessels present and sometimes quite a number, as in the two eyegrounds in Figs. 2 and 5, as well as the one shown in Fig. 8. That the conditions which we usually see are due, in a measure at least, to some sort of morbid processes, set up after the eyeball has been developed to something near its adult state there is, it seems to me, preponderating evidence. In nearly all cases the vessels of the retina are seen coursing over the defective space, and in some instances where the eye has come to a section, remnants of the retinal tissue have been found. Indeed Pause reports (*Graefe's Archiv*, xxiv, 2, 1878) a case where a perfectly normal retina was found over the entire extent of the coloboma. This would seem to show that the causes operating to produce the choroidal defect came after the time of the retinal development, and that they should in no sense be referred to a failure of the fœtal cleft to close. The formation of the retinal vessels does not take place until sometime after the cleft is closed, and their existence is therefore a proof that the primary vesicle has progressed normally in its development up to that point in the formation of the retina. The eyeground represented in Fig. 8 shows not only the proper distribution of the retinal vessels but also apparently normal

choroid forming a bridge across the coloboma dividing the space into two, and also a normal strip dividing these from the normal-appearing optic disc. It will be seen, too, that the coloboma does not extend to the periphery. There was associated with it a coloboma of the iris, likewise downward. The other eye was normal. Moreover, it is difficult to account for the atrophic choroidal spot B, in Fig. 5, on any supposition other than that it is the result of a choroiditis. There are vessels and remains of stroma and the pigmented edges which we are accustomed to find as the result of inflammation of the choroid. As to those colobomata of the choroid which have been ob-

FIG. 8.—Partial coloboma of choroid Remains of choroidal stroma and vessels in the defect. O. D. and retinal vascularization normal

served in other parts of the background, including those interesting cases of coloboma of the macula (one of which I have observed and reported in *Knapp's Archives*, Vol. XI, No. 4) it would indeed be hard to account for them on the supposition of a non-closure of the cleft, unless the idea suggested by Pflüger be accepted that the eyeball rotates after the closure, of which there is not, otherwise, the least evidence.

Lindsay Johnson suggests that colobomata at the macula are due to nævi of the choroid which disappear leaving the atrophic spot. In fact, it seems reasonable to suppose that the evolutionary forces, particularly those concerned in the nutritive supply, are interfered with, not necessarily by the failure of the cleft to close, but by some of the other numerous causes to which such inhibitions are attributed—prominent among which, no doubt, are disturbances in the vascular supply. That the vascular system generally has deviated largely from the normal is also shown in the peculiar order of vascularization of the retina, and other changes, particularly the smallness of the arteries and the tortuosity of the veins. That these interferences, however, are more liable to take place in the region of the invagination of the primary vesicle, where so many evolutional forces must meet and har monize, has been already admitted. It must also be apparent that the condition of these tissues under these circumstances would be such as to render them very liable to morbific processes, such as inflammation, or some retrogressive or misdirected activities.

That some of these processes are set up after the eye has attained something near its normal size and form is shown conclusively, I think, in the fundus picture of the right eye (Fig. 2). There can hardly be a doubt that in this case the optic nerve at the period of its full development occupied very nearly its normal position and that its scheme of vascularization approached that which we commonly see. Later, however, an intraocular pressure working on a tissue of low vitality and weak structure, pushed the nerve head and surrounding parts backward, dragging upon and separating its fibers, especially the upper ones, and disturbing the relations of its parts and its vascular system and bringing about the conditions as we see them now. It will be observed that the lower edge of the nerve head is still clearly limited and shows none of the appearances which we should expect to see in the so-called coloboma of the disc. From these appearances I believe that the larger part of the distortion of this eyeground is due to pressure on delicate or not fully developed tissue. In this way we can account for most, if not all, of the features of the so-called *coloboma of the optic nerve and optic nerve sheath.* Here again we are brought up against the commonly accepted explanation of defects in the nerve head and sheath by the non-closure

of the fœtal cleft. The fœtal cleft, however, does not affect the optic nerve development any more than it does the evolution and growth of the iris. The optic nerve is developed by offshoots from the retina after the mesodermal envelop has closed round the primary vesicle; and, while the nerve occupies the place of the optic stalk, the latter takes no part whatever in its development and the fœtal cleft can affect it in no way. These defects in the nerve head have been found, too, affecting other parts than those in the direction of the fœtal cleft. They are almost as frequent lateral as vertical and they differ in extent from an unusually large normal excavation to that we have pictured in Fig. 3. They are sometimes seen independent of any other anomaly in development of the background of the eye.

In brief, then, we do not see how coloboma in the eye tunics can be due essentially to failure of the fœtal cleft to close; for, in neither the iris nor nerve is there, at any time, during their development a cleft or anything approaching it, and as for the choroid, the tissue from which it is developed (the mesoderm), is continuous over this region from the beginning. As, however, the region of the invagination of the primary optic vesicle is the meeting ground for several developmental forces, it is easy for a disturbance to occur in the harmony of their action, with an anomalous or defective process of some kind as a result. This, it would seem, is affected chiefly and primarily through some fault in the evolution of the vascular system. This arrested or defective development of the tissues renders them more liable to the influence of pressure and the inflammatory process, and it is to these two that we are to refer many of the appearances which form a part of the picture in nearly every case of coloboma, and of the choroid particularly.

ONSET OF ACUTE GLAUCOMA CHARACTERIZED BY ENORMOUS CONJUNCTIVAL CHEMOSIS, AND VERY RAPID FAILURE OF VISION. IRIDECTOMY. RECOVERY OF SIGHT.

BY S. C. AYRES, M.D., CINCINNATI, OHIO.

SISTER A., aged 54 years, while engaged in conversation during the evening recreation hour, felt a sudden pain in her left eye. It seemed as if a strong blast of wind had been blown in her face, and almost immediately she could only see dimly the faces of those around her, while a moment before she could see clearly. The pain was sharp and severe and in a few moments she could see scarcely more than the motions of her hands. There began at once also a chemosis of the ocular conjunctiva.

I saw her the next day and the eye presented the following appearance: The cornea was steamy, the pupil widely dilated, and the ocular conjunctiva so chemotic that it stood like a rim around the corneal margin. Vision was reduced to counting fingers at 10″. The tension was + 2 and the eye quite painful. So great was the chemosis that it was impossible to make an iridectomy, and hot fomentations and eserine were ordered.

The next day the eye was more comfortable and the pupil slightly under the influence of eserine; but vision not improved, and the chemosis was slightly better. Eserine and fomentations continued

On the next day the eye was better in all respects except the vision. The pain was less and the chemosis was subsiding.

On the following day—the fourth from the time of attack—the chemosis had sufficiently subsided so as to enable me to operate. A liberal iridectomy was then made upward. The wound healed kindly and recovery was prompt and satisfactory. Her vision is now as good as it was before the attack.

The fellow eye had a similar attack three years previously which was characterized by the same symptoms of chemosis and sudden blindness. It was successfully operated on by Dr Robert Sattler.

The particular interest in the case is the rapid development of chemosis attending the onset of the attack of acute glaucoma in both eyes. It resembled that of acute purulent conjunctivitis. It would have been impossible to have operated on her when I first saw her. The use of warm fomentations gave great comfort and the weak solution of eserine reduced the tension and produced some contraction of the pupil.

THE REASONS IN ONE STATE FOR A LAW MAKING CREDÉ'S METHOD FOR THE PREVENTION OF OPHTHALMIA OF INFANCY OBLIGATORY TO PUBLIC INSTITUTIONS.

BY LUCIEN HOWE, M.D., BUFFALO, N. Y.

AS A PAPER with a title very similar to this has recently been published in the "Transactions of the American Ophthalmological Society," there is naturally danger of repeating here much of what was said there. That paper, however, dealt with the question in the abstract. This takes as a concrete example the data on the subject obtainable in a single state, what the local evidence there, is in favor of the use of Credé's method, and on the other hand, how little attention is really paid to its use, especially in the country districts. In giving these data it is necessary to repeat, in the proper place, but very briefly, the results published by obstetricians of their experience with the Credé method. That part relates to the general aspect of the question. The rest, however, relates to the principal cause of blindness in New York State. Imperfect as this outline is, it indicates to what practical results such a study might lead, also in other states.

As to the number of blind in the State of New York, we know from the statistics of 1890 that the total then was 4,389.

We know, also, that purulent ophthalmia of infancy produces more blindness than any other cause, or indeed, than many causes combined. We know it sends about 21 per cent. of the pupils to the two schools for the blind on 34th Street in

New York and to the State School at Batavia. And we know that in general it produces nearly 11 per cent. of the blindness among persons of all ages; or, to be very moderate, counting it 10 per cent., we have in the State of New York 438 persons blind from ophthalmia of infancy.

It is important, also, to remember the cost of that disease to the State. Suppose that out of these 438 persons, 8 per cent. could support themselves—a number larger than the largest in any estimate—we still see the number to be supported is 403. As to the cost of these we have

For 64 pupils at the two schools mentioned .	$ 16,547.38
For 339 others, if all were paupers, . . .	42,375.00
Or, for the 403 the yearly cost is over . .	58,622.00

Again, it is proper to notice how the blind population is distributed throughout the State, for the bearing of this point will appear later.

In order to show this at a glance, a map has been made, based on the figures given in the United States census report of 1890. The point which it emphasizes is that blindness is *less* frequent near the larger centers of population than it is in rural districts. Although this is contrary to what one might expect apparently the reason is not difficult to find. For, as ophthalmia of infancy is everywhere by far the most important factor in the production of blindness, and if, as it appears, less care is taken to guard against that in the country than in the city, it is natural to expect a larger number of children to develop that disease in the former than in the latter. Moreover, if this unusually large number of infected eyes in the country can not receive as prompt attention as they can in the cities where transportation is easy, physicians abundant, and dispensaries supra-abundant, then it is an additional reason for the existing distribution of the blind

Having thus glanced at the number made blind by this disease, how much they cost, and where they probably are, let us see what is the relative value of different metbods of preventive treatment. It should be remembered that this is not a matter of opinion simply, but a question of recorded fact. A fact established by the combined experience of obstretricians. The best and the most recent tables of this combined experi-

ence of obstetricians in all parts of the world were published last year by Kostling, of Halle, in the *Archives für Gynäkologie*. This shows that in

17,767 births with no treatment, 9.2 per cent. had ophthalmia of infancy.
24,724 births with 2 per cent. solution of silver nitrate, 0.65 per cent.
1,223 births with 1 per cent. solution of silver nitrate, 2.4 per cent.
1,623 births with carbolic acid solutions, 7.7 per cent.
965 births with 0.1 per cent. of sublimate, 0 6 per cent.
1,396 births with other sublimate solutions, 0.4 per cent.
6,155 births with sterilized water, 2.8 per cent
701 births with iodide trichloride solutions, 1.2 per cent.

For this interesting table it is possible to give only two words of comment. One is to call attention to the fact that ophthalmia is about fifteen times less when silver nitrate is used than when nothing is used. The second comment concerns the apparent advantage of sublimate solutions. This advantage, howover, is more apparent than real. The stronger solutions of sublimate proving very irritating to infants and several obstetricians like Fleischhauer, Olshausen and Widmark, after using the sublimate solutions, returned again to the silver nitrate.

Lest there should be any fear about using this, it is proper to answer briefly one or two possible objections to it. Some fear it may be dangerous. That is not so. In spite of the 24,000 cases here recorded, in spite of the efforts to find bad results from the use of silver nitrate which Cohen made by letters from one hundred and ten of the largest German clinics, in spite of the search of reported cases everywhere and which would surely have been reported, it is possible to find only four questionable cases. For two of these children drops were ussd. No one knows of what. For one (de Schweinitz' case) the solution was 4 per cent. in strength. Another (Pomeroy's case) the child was probably a so-called "bleeder."

Again, the method is not especially painful if cocaine is used first, as I have found on the blind eye of an adult, and especially is the discomfort not so great if we follow the suggestions of Dr. Alleman by using the fused silver nitrate for the solution.

This is the evidence of statistics. Let us come near home to ask what experience, not published, has taught obstetricians here as to the point in question.

About a year ago I wrote to most of the leading members of the New York Obstretrical Society asking: "Should Credé's method be invariably used in public institutions, and shonld it be made obligatory there?"

Out of thirty-three replies received, twenty-eight answered the first question by "yes," and twenty-three answered the second question by "yes." The following have said that it should be made obligatory, namely: Drs. Boldt, Brettauer, Byrne, T. W. Cleaveland, Clement Cleaveland, Coe, Currier, Gibb, Emmett, Goffe, Jarman, Jacobi, Marx, McLean, Morrill, Mundé, A. M. Thomas, T. G. Thomas, Tucker, Tuttle, Vineberg, von Ramdohr, and Wiener. So it is evident as to what should be done.

Next let us inquire what is actually done. Comparatively little. In order to ascertain this, I have written to the principal hospitals and lying-in asylums and to the physician-in-charge of the alms-houses in the sixty counties.

An entire paper could be devoted to their answers. The points which concern us, however, are, first, that the leading obstretricians, especially in large cities, employ Credé's method almost without exception. In the alms-houses, however, it is almost invariably omitted. This has an instructive bearing on the may before referred to.

The foregoing facts show that the number of blind from this disease is large; that its cost is great; that the combined experience of obstetricians as published by observers in different parts of the world point to one remedy as much better than any other thus far known. A remedy that is safe, easy of application, and if generally used, would greatly reduce the number of blind and consequent cost to the State. The statistics alone indicate the desirability of having that treatment made compulsory. In addition, however, there are other reasons not statistical in favor of such a law. There are:

1. That when the State assumes the care of a child by supporting it, the child becomes practically its ward, in that case the State, as the guardian, has the right to dictate what treatment shall be employed for the child, and if one method of treatment has been proved by vast experience to be superior to every form of treatment thus far known, then the State has the right to demand that any physician treating such a child shall use that form of treatment which insures the greatest amount of safety.

2. In the same manner it is not only the right but it is also the duty of the State to demand such treatment for pauper children, this being not simply for the sake of the child, but also to relieve the State from heavy, and to a great extent unnecessary, taxation.

3. The State has already established this principle of compelling physicians to follow certain procedures. not only by obliging them to report contagious diseases and imposing other duties upon them, but especially has it done so by the enforcement of vaccination. In this it has furnished a precedent for compelling physicians to use a certain method of treatment to prevent a certain disease.

It is true that a remedy better than silver nitrate may be discovered any time, but if this happened to-morrow the law could easily be amended. It has required nearly twenty years to accumulate the present evidence on this point, and while we wait for some other discovery, each year more children are made blind for lack of such legislation.

It is also true that such legislation would restrict the action of the few physicians employed by the State when they are attending the obstetric cases in alms-houses, but in view of the advantages certain to follow from this legislation, it is not probable that the medical profession would oppose such a saving and humanitarian effort when its real purpose is understood.

For it is simply a question as to whether a point of professional pride shall be maintained or whether by yielding that, there shall be each year a great saving to the State together with the rescuing of a few, perhaps a considerable number of children from life-long blindness.

The result of such a law would be:

First—That it would immediately tend to lessen the number of children thus affected.

Second—The indirect moral effect would be good in sustaining practitioners who do use this method in spite of ignorant objectors.

Third—The moral effect would be good in condemning obstetricians who neglect its use by such omission run greater risks of adding to the number of blind, nearly every one of whom, whether psupers or not, was educated at the expense of the State.

EXPERIENCES DURING THIRTY-EIGHT YEARS OF OPHTHALMIC PRACTICE WITH LARGE PARACENTESIS OF THE SCLEROTIC WITH CILIOTOMY IN ACUTE GLAUCOMA (MR. HANCOCK'S METHOD).

BY S. POLLAK, M.D., ST. LOUIS, MO.

THE following is a plea for a mild, quick and effective surgical procedure for the relief of pain in acute glaucoma, and with a final outcome far more satisfactory, than has been heretofore attained. Thus, this short paper is presented with the object of recalling to the ophthalmic surgeons an operative method hitherto either not recognized or neglected by the profession and with the hope of eliciting a discussion thereon, advantageous to both the surgeon and patient.

It is not necessary to enter nosologically upon the symptomatology, pathogenesis and etiology of glaucoma, they are well known to every practitioner of medicine. The syndrome of glaucoma is so striking that it can be recognized at the first glance. All agree that the increased tension of the eye, by excess of secretion or diminished excretion, or both, is the chief if not the only cause of this most painful of eye diseases. To abstract the exudate, and re-establish the normal in- and outflow of the fluids, is the main if not the only object of treatment; but this can not be reached by any pharmaceutical remedy, and must, therefore, inevitably come into the domain of ophthalmic surgery. *Tapping* of the cornea, one or more times, with a broad paracentesis needle effects it in part, but only temporarily, for the withdrawal of the fluid contents of the eye is never complete, the punctures close too quickly for the normal opening of the natural drainage channels; the morbid hypersecretion commences again, and the tension becomes as great as ever, with all the attending consequences.

In 1856 the illustrious von Graefe announced a radical cure of glaucoma by means of a *large iridectomy*. The authority of that famous man was sufficient to attract universal attention, his opinion was law to the ophthalmic world. His advice was strictly and implicitly followed by oculists everywhere.

In the winter of 1859-60 I attended the clinics of von Graefe with absolute regularity, and saw a great deal of his work. I was charmed with and edified by the brilliancy of his eloquence, fervor and earnestness in his lectures, even more so than by his practical work. From Berlin I went to Paris, there von Graefe was not a great favorite, and his teaching was not accepted without protest. In Vienna von Graefe's precepts stood nearly as high as in Berlin, yet they were occasionally excepted to and varied from. It was in London where he exercised autocratic powers. At Moorfields—the greatest of all eye clinics—a large iridectomy in glaucoma was honestly performed, with the only difference, that a broad curved lance knife was used, instead of the narrow-bladed cataract knife of von Graefe. The entrance into the anterior chamber was made at the upper sclero-corneal junction.

In this most magnificent clinic, with a large staff of the most able ophthalmic surgeons and world-renowned medical scientists, such as Bowman, Critchett, Hutchinson, Hulke, Streatfeild and Bader, where clinical material was superabundant, furnishing cases for ophthalmic surgery of every kind and great variety, glaucoma was, perhaps, of more frequent occurrence than in the United States. Von Graefe's plan of treatment of glaucoma by large iridectomy was faithfully carried out but I did not have the opportunity of seeing the patients weeks after treatment.

From Moorfields I drifted daily to the eye clinic of Charing Cross Hospital, in charge of Mr. Hancock, as oculist-in-chief. I saw before me a very quiet, unassuming, unpretentions, but most courteous gentleman who went about his work quietly, without any eclat and without elocutionary display. He had three very able coadjutors, very well trained, to do all clinical work in prompt, precise and well-regulated manner. No time was wasted in vain verbose discussion. A diagnosis once made was promptly acted upon, according to well-established rules. A military precision prevaded every department of the clinic. When a case of acute glaucoma was brought up he simply addressed a few words to the numerous visitors, only stating that he assumed that they were familiar with the pathology of it, and with the therapeutic measures required. "We must reduce thoroughly the tension, and I propose to do it by a quick and effective method, and this is by a *large paracente-*

sis of the sclerotic with ciliotomy." His aids were prepared for that statement, and the operation was performed ere the patient was aware of what was to be done. Usually it was done while the patient was sitting up in a chair. Only the very nervous were allowed a recumbent position. This procedure was certainly quite different from one which I had seen done, perhaps, only a few hours before, at Moorefields, and struck me, and the others present, as greatly preferable and certainly more satisfactory.

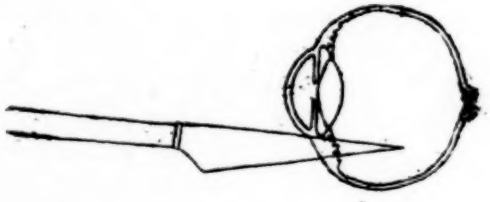
I will briefly describe and compare the two methods:

1. THE VON GRAEFE METHOD: LARGE IRIDECTOMY. — After deep anæsthesia with chloroform — cocaine was not known then—a speculum is placed into the palpebral fissure, the eye is fixed and kept immovable with a long-toothed ophthalmostat, an opening is made in the upper third of the sclero-corneal junction with a narrow-bladed cataract knife, in order to enter the anterior chamber, and pass through its entire width in order to make the counter puncture This is, however, very difficult, and sometimes impracticable. The anterior chamber is apt to be very shallow, sometimes even entirely obliterated, the iris lying close to the cornea, the knife can not be passed between without wounding the iris, causing hæmorrhage and obscuring the field entirely. After the counter puncture has been made, corneotomy has to be completed by a to-and-fro or sawing motion of the knife held edge upwards, but this manœuvre causes a tugging on the fixation forceps and laceration of the conjunctiva. The iris is then seized with iridectomy forceps, is incised and at least one-fifth is *torn* off from its peripheral or ciliary attachment, and finally cut off. This is exceedingly painful as is shown by the flinching of the patient in spite of the deep anæsthesia. The anterior chamber is full of blood, which sometimes coagulates, and can only be cleared by absorption after many days. The eye is then bandaged and the patient put to bed. The fluid contents of the eye having escaped, the tension is reduced, and relief is given from pain. A goodly portion of the secreting surface has been removed, the resulting cicatrix being of very thin tissue, facilitates subsequent filtration; the laying bare of the zonula Zinii so near up to the ciliary insertion, improves also the interchange of fluids between the vitreous and aqueous humors, thus the recurrence of the glaucomatous features may be prevented.

But a cystic cicatrix is frequently the result, which is permanent in character and causes great irritation by the movements of the lids. Besides, the coloboma of the iris is perpetual, and so is the mutilation, marring the eye cosmetically and functionally.

Even in the skilled and dexterous hand of von Graefe at least twenty minutes are required for the performance of this most painful operation, and many weeks of most careful nursing, ere the final result can be ascertained.

2. HANCOCK'S METHOD: LARGE PARACENTESIS OF THE SCLEROTIC WITH CILIOTOMY.—No anæsthetic required on account of the celerity of the operation. The cornea being insensitive, a speculum is dispensed with. As the eye is usually turned up, fixation forceps are not needed. The lids are separated are kept open by the thumb and index finger which steady the eye at the same time. A Beer's knife held edge downward is thrust into the sclerotic at the lower margin of the cornea and carried slightly *downward* and backward into the vitreous to half the length of the knife, by which a large incision is made in the sclerotic, the ciliary muscle divided, and the vitreous body pierced. In withdrawing the knife, it is



almost imperceptibly turned to one side, thus making the wound gap, and giving an outlet to the entire fluid contents of the eye, and even to a little of the vitreous fluid. A few seconds only are required for this operation. No dressing or any after-treatment is needed. The relief from the increased tension and the pain is instantaneous. The lens recedes to its normal position, the pressure upon the ciliary processes is removed, the iris being freed from pressure soon resumes its normal place, the spaces of Fontana are gradually opened and so is also the canal of Schlemm. Drainage through the freed normal channels is now made possible and soon re-established. A small leakage through the wound can be kept up as long as

desirable, so that a return of increased tension is not likely to ensue. The eye retains its normal appearance, the pupil assumes its normal size. The turbid exudate having been entirely evacuated, the cornea and lens soon clear up, and vision, if not destroyed before the operation, is soon partially regained.

The Hancock method was well known as Moorfields, but was never alluded to or practiced. When I inquired of Mr. Critchett the reason for this, he tried to avoid an answer, and I did not press it. I knew that jealousy prevailed in the London Hospitals as much as elsewhere, but neither praise nor criticism was passed on each other.

On my return to the United States I opened an eye and ear clinic in the "St. Louis Mullanphy Hospital." The clinic soon grew in favor and developed into a very extensive charity. Clinical material was abundant and ophthalmic and aural work was carried on honestly and fairly, with a reasonable degree of success.

Chronic primary glaucoma was frequently seen, and treated therapeutically by constitutional means, such as mercurials, iodide of potassium, salicylate of soda, locally, by pilocarpine, and later by eserine, occasionally by local abstraction of blood, and finally by tapping the cornea.

The first five cases of *acute glaucoma* I iridectomized, but with unsatisfactory result, either to myself or patients. The procedure was slow, very difficult, and very painful, requiring always a protracted after-attendance, leaving a mutilated eye with poor and painful sight, or no sight at all. Such was the prestige established by von Graefe, that I had not the courage to give up the large iridectomy or deviate in any manner from the rules laid down by him; I became impressed with the fear lest, in case of my failure, I would not be sustained by the profession. But the unsatisfactory ending of the fifth case determined me to give up the von Graefe method and substitute that of Hancock's for it. It proved to be a happy and most fortunate resolve. In the *thirty-eight years* of my ophthalmic work *I have performed large paracentesis of the sclerotic with ciliotomy between sixty and seventy times without a single failure.* It gave immediate relief from pain, arrested effectually the progress of the disease, and I never met with a recurrence of it in a single instance. Many of the patients were seen weeks, months, even years after the operation; the cure was

found to have been radical and permanent. One patient came expressly all the way from New Mexico with a threatened acute glaucoma of *the second eye* four years after the other had been operated upon. Knowing what was coming, he lost no time and *demanded* that the operation be performed forthwith, as his business required his constant presence. His demand was reasonable and *à propos*. It was promptly complied with. He left the city on the next train. I only heard from him several months later, by his sending another patient with a letter of introduction.

I have the clinical record of all the cases of acute glaucoma, which I have treated, by either the von Graefe or Hancock's method, in hospitals or private practice. They are occasionally enquired into by pension agents or pension commissioners. Some of these patients have called many years after, for optical treatment, but never for a recurrence of glaucoma. Below will be found a tabulated statements comparing the salient points of the two operations in my practice:

	Large Iridectomy.	Large Paracentesis of Sclerotic With Ciliotomy.
1.	Deep anæsthesia needed.	No anæsthetic required.
2.	Five or more instruments needed.	Only one instrument necessary.
3.	Operation difficult, very painful.	Operation easy and almost painless.
4.	Duration of operation 15–20 minutes.	Duration of operation 5–10 seconds.
5.	Drainage effective but hæmorrhagic.	Drainage effective without hæmorrhage.
6.	After-treatment, protracted.	No after-treatment at all.
7.	Final result, mutilated eye, damaged in appearance and function.	Final result, no mutilation, appearance and function normal.
8.	Recurrence rare.	Recurrence never.

I shall conclude this paper with a reference to the last case of acute glaucoma which came under my notice:

B. G., aged 35 years, a very decrepit, anæmic, prematurely aged woman, looking at least 50, presented herself on January 17, 1898, with the typical well pronounced symptoms of acute glaucoma of the right eye with turgescent tortuous blood vessels of the sclerotic, chemosed conjunctiva, hazy, smoky, insensitive cornea, dilated pupil, and a very shallow anterior chamber. The bulbus was of marble hardness with turbid media and opalescent lens; fundus not illuminable, and mere perception of light. She suffered excessive pain in the right eye, in

the temporal, supra-orbital and occipital regions, preventing sleep, and causing great general malaise. The irides of *both* eyes were tremulous, probably due to synchysis, or to ectopic dislocation of the lens.

An immediate operation was proposed, but refused.

Driven by the severe and continued pain, she returned to the clinic on January 20, ready and willing to submit to the proposed treatment.

In the presence and with the valuable assistance of my friend and associate, Dr. J. Ellis Jennings, and without much ado, and without an anæsthetic, except one drop of cocaine, a large paracentesis of the sclerotic with ciliotomy was performed in about ten seconds. The escape of aqueous and vitreous fluid was rapid and complete, followed by a total relief of pain and subsidence of all prominent symptoms of glaucoma. A very little absorbent cotton was loosely put upon the eye, without any bandage, and the patient sent to bed, to enjoy the quiet repose of the righteous and well.

Next day the pupil was smaller and the anterior chamber forming. The fundus not yet illuminable on account of a very slight hæmorrhage in the vitreous; no sight.

Third day, red reflex seen, with perception of light.

Fifth day, fundus seen, sight much better.

Seventh day, counts fingers at 20 feet, reads large print; anterior chamber fully formed, iris in its place, pupil yet a little dilated, tension normal. She feels well, with an eye in appearance and functions like its mate.

She was carefully examined by Drs. Alt and Jennings three weeks after the operation, who promised to add a few remarks on this wonderfully rapid and complete result.

It is my firm belief that acute glaucoma will be bereft of the terror it causes to the oculist and patient, by the Hancock plan of treatment, which is hoped will soon be adopted generally.

Thanks to Dr. Jennings, I am able to add a few lines of the literature on "Intra-Ocular Myotomy or Hyposcleral Cyclotomy."

This operation was performed on the supposition that spasm of the ciliary muscle causes a stasis in the intra-ocular blood circulation.

It was performed by Hancock in 1860, Heiberg in 1862, Pritchard in 1871, and Detowski in 1872.

Hancock inserted Beer's knife at the outer lower scleral border and passed it down and back till the sclera showed a 3 mm. section; or after inserting it at the limbus and through the angle of the anterior chamber, the muscle was cut with the lens on one side and the sclera on the other side of the knife.

Autopsies showed that many of these incisions had divided a part of the muscles.

But there is a sequel to the above case. On February 15 B. G. called again at the clinic stating, that on rising the day before she perceived that the right eye was sightless and painful, which was unfortunately too true, but readily accounted for. As stated before that there is and always was a tremulous iris in both eyes, in the left even more so than in the right. This was attributed to *synchysis* and probably to *partially dislocated lenses*, which, however, was not made out by ophthalmoscopic examination. But at present there is *a complete dislocation of the lens into the anterior chamber of the right eye*, causing much pain in the ciliary region and considerable increase of tension. The lens is opalescent, though it was clear the day before. She met with no injury of the eye which could have caused a rupture of the suspensory ligament and a consequent displacement of the lens. It is clear that the *partial dislocation* of the lens, which has existed so long, has now *developed* into a *complete dislocation* in the right eye, as is now very manifest, and has doubtless caused the present aggravated condition. It is also more than probable, that the long existing partial dislocation of the lens may have been the initial cause of the glaucoma. According to Mr. Bowman this is due to the pressure of the lens upon the iris, which sets up irritation and a hypersecretion of fluid within the eye.

The exigencies of the case were such, that the lens had to be removed. She returned on the 17th for that purpose. The lens was transfixed with a cataract needle, a lower one-third section of the cornea was made with Beer's knife and finished with a conjunctival flap. With a Waldau scoop fragments of the lens were drawn out, until the pupil became clear and she counted fingers. The corneal wound was carefully closed, the conjunctival flap was smoothly adjusted over it, a few drops of eserine instilled, the lids closed, and a pledget of a few layers of aseptic gauze laid over them and fastened with a few strips of isinglass plaster. She was sent to bed, is entirely free from pain, slept good, and is in the best of spirits.

A FEW WORDS IN ADDITION TO DR. POLLAK'S PAPER.

BY ADOLF ALT, M.D., ST. LOUIS, MO.

ON FEBRUARY 8, by the kindness of Dr. S. Pollak, I had an opportunity to examine the eye of B. G., operated by him after Hancock's method some days previously. At his instance I wish shortly to state the conditions found.

The right eye showed no injection. There was a semi-transparent linear scar beginning at the corneo-scleral margin of the lower inner quadrant and running down in the sclerotic for about 5 mm. on the nasal side of the inferior rectus. The anterior chamber was fairly deep. Iridodonesis was well marked (in both eyes) Tn.

The ophthalmoscope showed numerous floating opacities. In the neighborhood of the macula lutea were two glistening white spots without a pigment rim, between them some signs of former choroiditis. (In the fellow eye a similar condition existed). The optic nerve and blood vessels were normal. Vision in R. E., $^{5}/_{CC}$, with + 6 D., $^{20}/_{LXX}$.

On February 15, I again saw the patient, with total dislocation of the lens into the anterior chamber.

It seems to me evident, that the defects in the choroid and zonule of Zinn were congenital. The glaucoma in this case, although acute, was a secondary glaucoma. In fact, the patient stated, that the attack for which she had been operated on had started exactly in the same manner by dislocation of the lens into the anterior chamber.

Nevertheless, the operation after Hancock's manner had undoubtedly done all that Dr. Pollak claims for it, relieved the acute attack and the pain accompanying it, had led to restoration of a fairly deep anterior chamber, and $^{20}/_{LXX}$ of vision.

Whether this method can be applied with equal success in all cases of acute glaucoma, is a question which, it seems to me, time has already decided While iridectomy to this day is practiced by almost all surgeons, Hancock's operation is forgotten by most of the older operators and unknown to the younger ones. Whether it will be worth while to revive it, the future will, perhaps, show.

ADDITIONAL NOTES ON A CASE OF HIGH MIXED ASTIGMATISM.

BY B. L. MILLIKIN, M.D., CLEVELAND, OHIO,

PROFESSOR OF OPHTHALMOLOGY, WESTERN RESERVE UNIVERSITY, — OPHTHALMIC SURGEON TO LAKESIDE HOSPITAL, — CONSULTING OPHTHALMOLOGIST TO CHARITY HOSPITAL, CLEVELAND, OHIO.

IN THE "Transactions of the American Ophthalmological Society, Vol. VI, page 582, I have reported in detail the history of a case of very high mixed astigmatism, the correction at that time being as follows:

O. D., — 10.00 D. cy. ax. 90° ⊂ + 7.00 D. cy. ax. 180°.

O. S., — 6.00 D. cy. ax. 100 ⊂ + 4.00 D. cy. ax. 10°.

These glasses were prescribed in 1891. Since then the patient, who is a book-keeper, had worn the glasses steadily until December, 1896. For six months before this time, however, he had complained of some blurring, both for distant and for close use of the eyes.

Examination on December 7, 1897, gave the following results: With his old glasses his vision was $^{6}/_{XVIII}$ in each eye. A fresh examination of his refraction developed the following requirements in the way of glasses:

O. D., — 13.00 D. cy. ax. 95° ⊂ + 7.00 D. cy. ax. 5°.

O. S., — 8.50 D. cy. ax. 100° ⊂ + 5.00 D. cy. ax. 10°.

These glasses were prescribed for distant use. One week later, when his glasses were ready, it was found that his vision with the right eye was $^{6}/_{IX}$ full, and with the left $^{6}/_{VI}$. In testing his near vision it was found at this time that he was somewhat presbyopic. With + 2.00 D. added to each eye he was able to read perfectly well at 14 to 15 inches, and these glasses were ordered for close use over the distant ones.

Since then the patient has made no report, so that I have no doubt he is using them for all sorts of work with comfort.

An examination of the eye-grounds, with the glasses on, showed the fundus of each eye in very good condition, with no evidence of any marked disease. In fact, there had been practically no change in the eye-grounds since the primary examination in 1891.

It will be observed that in the glasses the increase of the

myopic portion of the right eye had been from 10.00 D. to 13.00 D., with no change in the hypermetropic cylinder, as compared with the previous examination in 1891, while in the left eye there had been an increase from 6.00 D. to 8.50 D. in the myopic cylinder, and an increase from 4.00 D. to 5.00 D. in the hypermetropic cylinder, a difference in the right eye of 3 00 D., and a difference in the left of 3.50 D. in a period of five years. It will be seen, therefore, that the total astigmatism reaches the very high amount of 20.00 D. in the right eye and 13.50 D. in the left, with practically normal vision. It will also be observed that the angles of the astigmatism are all against the rule.

Thus far I have been unable to find any case of so high a degree of astigmatism with practically normal vision and the ability to use the eyes for all sorts of close work without discomfort.

CORRESPONDENCE.

INVITATION TO A SUBSCRIPTION FOR A MEMORIAL TO OTTO BECKER.

VIENNA AND ERLANGEN, February, 1898.

OTTO BECKER unfortunately did not reach a great age. When he was 62 years old, possessed of good health and mental vivacity, filled with the desire to serve science, educate pupils and to help suffering humanity, an insidious disease attacked him and tore him from our midst in a few weeks.

His name will remain unforgotten in ophthalmology. Yet, as his 70th birthday anniversary is approaching, we desire to celebrate his memory by a visible and lasting token.

In the Domhof at Ratzeburg there stands the small house in which Becker was born. We wish to ornament this with a memorial tablet. At the same time we want to place the bust

of the master in the place where he worked for almost twenty-five years, in the beautiful eye clinic at Heidelberg, which owes to him its origin, to his organizing talent its excellent arrangement, and to his prominence its name and fame.

We have placed the execution of a life-size bronze bust into the hands of the well-known artist, Professor Koenig, at Vienna. It will probably be finished by July of this year.

We confidently address all pupils, friends and colleagues of Otto Becker, all members of the ophthalmological society of which he was so long an honored member, being satisfied that every one will gladly, according to his means, help us in this work of gratitude.

Contributions to be sent to

DR. ST. BERNHEIMER, Vienna, IX, Guentherstr. 3,

Or, PROF. O. EVERSBUSCH, Erlangen, Glueckstr. 10,

The Committee.

While the editor of this Journal does in no way wish to detract from the praise which so deservedly was and is still heaped on Otto Becker, and hopes that the American colleagues will gladly respond to the request, he nevertheless, having been a pupil of both, thinks it no more than just to remind the Committee that in order to honor the dead, the living should not be robbed of his due. It was Hermann Knapp, now of New York, who founded the eye clinic at Heidelberg, and who had brought it to name and fame without Government aid, while Otto Becker was still an assistant at Vienna. It is sometimes well to see to it that history is not turned into legend too soon. When Becker came to Heidelberg in 1869, Knapp's clinic was bought by the Government and turned over to him a ready-made, well-arranged and deservedly renowned clinic, the creation of the untiring energy, scientific worth and practical accomplishments of Herman Knapp.

ALT.